Intrauterine Fetal Resuscitation

Patricia M. Witcher, RNC-OB, MSN
Northside Hospital
Atlanta, GA
Traditional Interventions

- Lateral positioning
- IVF bolus
- Reduction of uterine activity
  - Decrease/discontinuation of oxytocin
  - Terbutaline
- $O_2$ by face mask
- Delivery
Intrauterine Fetal Resuscitation

- Ascertain and direct interventions based upon underlying pathophysiology
- Maximize maternal cardiac output
- Increase oxygen content in the blood
- Relieve umbilical cord compression
Fetal Oxygenation

- Fetal pO$_2$ much lower
- Fetal pCO$_2$ much higher
- Blood flow regulates pO$_2$ and pCO$_2$
Fetal Oxygenation: Influencing Factors

- Blood flow
- Oxygen content
- Placental factors
- Fetal circulation
DO₂ = CO x [(1.39 x Hb x SaO₂) + PaO₂ x 0.003)] x 10 dl/L
Benefits of Oxygen

- Debated\textsuperscript{1-5}
- Higher $\text{FiO}_2$ required to increase fetal $\text{SpO}_2$
  - Small increases in fetal arterial $\text{O}_2$ content and $\text{SaO}_2$ occurred with small increase in fetal $\text{PaO}_2$
- Reperfusion $\rightarrow$ ROS $\rightarrow$ oxydative stress $\rightarrow$ cellular damage (\: limit duration of $\text{O}_2$)
- Discontinue $\text{O}_2$ when FHR pattern resolves\textsuperscript{7}

\textsuperscript{1}Khaw, Lee.  Curr Opin Anaesthesiol, 2004; 17
\textsuperscript{2}Dildy et al.  Am J Obstet Gynecol, 1994
\textsuperscript{3}Fawole & Hofmeyr.  Cochrane Database Syst Rev, 2003; CD000136
\textsuperscript{4}Haydon et al.  Am J Obstet Gynecol, 2006; 195
\textsuperscript{5}Simpson & James.  Obstet Gynecol, 2005; 105
\textsuperscript{7}Garite & Simpson, Clin Obstet Gynecol, 2011; 54(1)
Determinants of Umbilical Venous pO2

(in the absence of maternal hypoxia or anemia)

Uterine blood flow

Maternal oxyhemoglobin dissociation curve

$\text{SaO}_2$

$\text{PaO}_2$
Fetal Oxygenation

- Increased affinity of fetal hemoglobin for oxygen
- High blood flow to fetal tissues
- Increased oxygen extraction
## Supplemental Oxygen

<table>
<thead>
<tr>
<th>O2 device</th>
<th>( \text{O}_2 ) Concentration</th>
<th>Flow Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal Cannula</td>
<td>24 - 44%</td>
<td>1 - 6 L/min</td>
</tr>
<tr>
<td>Simple Mask</td>
<td>35 - 55%</td>
<td>5 - 12 L/min</td>
</tr>
<tr>
<td>Partial Rebreather</td>
<td>35 - 60%</td>
<td>8 - 15 L/min</td>
</tr>
<tr>
<td>Nonrebreather</td>
<td>60 - 90%</td>
<td>8 - 15 L/min</td>
</tr>
</tbody>
</table>
Case Studies

Case studies are not provided in the handout material in order to protect the confidentiality of the patients and institutions from which they are derived.
Maternal Oxygen Delivery (DO$_2$)

Cardiac Output

Oxygen Content

Preload

Afterload

Contractility

Heart Rate

Hemoglobin

SaO$_2$

PaO$_2$

\[
DO_2 = CO \times \left[ (1.39 \times Hb \times SaO_2) + PaO_2 \times 0.003 \right] \times 10 \, \text{dl/L}
\]
Maternal Dependence Upon Cardiac Output

- Increased metabolic demands
- Increased $O_2$ consumption
- Increased $CO_2$ production
- Increased functional residual capacity
- Increased minute ventilation

Dependence Upon Cardiac Output
Maternal Cardiac Output = Fetal Oxygenation

- Increased cardiac output
- Uteroplacental perfusion
Lateral Positioning

- Alleviates vena caval compression
- Increases preload
- Increases CO
- Increases uteroplacental perfusion
IVF Bolus

- Increases circulating blood volume
- Increases maternal cardiac output
- Increases uteroplacental perfusion
Physiologic Adaptations that Accomodate Pregnancy

↑ cardiac output
↑ blood volume
↓ SVR

↑ uteroplacental perfusion (10% of CO)

Maximal uteroplacental vascular dilation
Placental Factors

- Placental surface area
- Thickness of surface membrane - fetal capillaries in the placental villi
- Placental vascular resistance
Effect of Uterine Contractions on Fetal Oxygenation

Elevated Uterine Activity and Risk of Fetal Acidosis at Birth

- Those with acidosis demonstrated shorter relaxation time, higher amplitude, and increased uterine activity.

Physiology: Autonomic Control of FHR

- Umbilical cord compression
- Uteroplacental blood flow
- Carbon dioxide retention
- Hypertension
- Hypoxemia / Acidemia
- Late decelerations
  Variable decelerations
  Prolonged decelerations
  Bradycardia
Uterine Activity

Uterine contractions

↑ placental vascular resistance

↑ impedance to blood flow

hypoxic episodes

Rapid redistribution of oxygen during contraction / return of blood flow between contractions

Acid-base balance maintained

Decreased ability to react to hypoxic episodes, reduced defenses or sustained reduction in blood flow

Hypoxemia and/or acidosis
Reduction of Uterine Activity with Beta Agonist

• Compared with no treatment, fewer failed improvements in FHR abnormalities with tocolytic agent

• Betamimetic
  • Reduced uterine activity
  • Not enough evidence to evaluate use for suspended “fetal distress”

Changes in Fetal Mean Cerebral Oxygenation

Progression to Passive Second Stage to Expulsive Efforts

Maternal Pushing

Increased intrauterine pressure

Valsava

↑ intrathoracic pressure

↓ venous return

↓ ventricular filling

↓ cardiac output

↓ uteroplacental perfusion

↑ oxygen consumption
"Nonreassuring FHR? Is it Mom?"

- ↓CO
- ↓uteroplacental perfusion
- ↓PaO$_2$
- ↑PaCO$_2$
- FHR abnormality
- Placental vasoconstriction
Case Study

Case studies are not provided in the handout material in order to protect the confidentiality of the patients and institutions from which they are derived.